Journal of Forensic and Legal Medicine 16 (2009) 59–63

Contents lists available at ScienceDirect

Journal of Forensic and Legal Medicine

journal homepage: www.elsevier.com/jflm

Original Communication

Qualitative and quantitative EEG abnormalities in violent offenders with antisocial personality disorder

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ARTICLE INFO

Article history:
Received 17 March 2008
Accepted 16 August 2008
Available online 1 October 2008

Keywords:
Electroencephalogram
Antisocial personality disorder
Violent offender
Quantitative EEG analysis

ABSTRACT

Resting eyes closed electroencephalogram was studied in a group of violent offenders evaluated at Psychiatric Department of the Legal Medicine Institute in Cuba (18 with antisocial personality disorder, ASPD, and 10 without psychiatric diagnosis). Characteristics of the EEG visual inspection and the use of frequency domain quantitative analysis techniques (narrow band spectral parameters) are described. Both groups were compared to Cuban normative database. High incidences of electroencephalographic abnormalities were found in both groups of violent offenders. The most frequent were: electrogenesis alterations, attenuated alpha rhythm and theta and delta activities increase in the frontal lobe. In the quantitative analysis theta and delta frequencies were increased and alpha activity was decreased in both groups. Differences appear for the topographical patterns present in subjects of both groups. EEG abnormalities were more severe in ASPD than in control group. Results suggest that EEG abnormalities in violent offenders should reflect aspects of brain dysfunction related to antisocial behaviour.

1. Introduction

Antisocial personality disorder (ASPD) is a relatively common psychiatric diagnosis. Prevalence estimates in the general population are 3% for men and 1% for women (DSM-IV-R, American Psychiatric Association, 2000).[1] This disorder is associated with a pervasive pattern of disregard for and violation of the rights of others, that begins in childhood or early adolescence and continues into adulthood, and not surprisingly, the highest prevalence rates of ASPD are found in prisons and forensic settings.[1] Several studies indicate an interaction between biological factors and social factors in the development of antisocial behaviour.[2–7]

In the last years there has been an increased interest in studying the neurobiology of personality disorders[8–10], and in particular ASPD.[11,12] The brain regions more compromised in antisocial populations include frontal and temporal lobes.[4,13,14] Functional alterations in these regions have been related with different types of violent behaviours while temporal lobe dysfunction may be associated with sexual offending, frontal lobe dysfunction has been claim to be associated with non-sexual violent offending.[15]

Neuroimaging studies have shown the involvement in this pathological condition of prefrontal areas, especially orbitofrontal cortex, and amygdala.[16–20] Also, impaired serotonin (5-HT) neurotransmission has been implicated, since subject with ASPD present alterations in measures of 5-HT system, such as blunted hormonal response to 5-HT pharmacological challenges and reduced 5-HT receptors numbers.[21–25]

A large number of studies have found EEG abnormalities in violent offenders. Hill and Pond (1952) and Bach-y-Rita et al. (1971) examined large samples of violent offenders and observed EEG abnormalities in about 50% of the subjects.[26,27]These findings have been replicated by other studies of murderers and other types of violent offenders.[16,28–31] One of the most frequently observed EEG abnormality consists of excessive slow wave activity. Whereas earlier studies were generally more qualitative, EEG technology has become increasingly more advanced, allowing for detailed quantitative computerized analyses instead of clinical visual inspection.

EEG studies of antisocial groups have become less common over the last several years. The reason has been due to the development of more advanced functional brain imaging techniques such as PET and fMRI that has begun to dominate the field. These techniques provide better spatial resolution and allow for the examination of specific subcortical structures, but there is evidence that EEG is sufficiently sensitive to detect differences when comparing these subjects with controls, even in relatively small sample sizes.[31,32]

However, the increasing information of neurological impairment in antisocial individual, very few studies have been conducted specifically to assess abnormal findings by means of quantitative EEG analysis in subjects with this disorder. For this reason the aim of this study was to investigate whether visual

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doi:10.1016/j.jflm.2008.08.001

and quantitative analysis of EEG could differentiate violent offenders with ASPD, and those without this psychiatric diagnosis and to quantify the nature of these differences. It is hypothesised that subject with ASPD will have increased theta activity, with reduced levels of alpha compared to normal subjects, and that violent offenders without this disorder have similar abnormalities but at a lesser degree.

2. Methods

2.1. Subjects

The study included 28 males who underwent forensics evaluation at the Psychiatric Department of the Legal Medicine Institute of Cuba for having been authors of criminal acts during the period of January 2002 to December 2005. Written informed consent was obtained from all subjects prior the study. This study was approved by the Ethics Committee of the Legal Medicine Institute.

The experimental group comprised 18 violent offenders with ASPD according to the DSM-IV-R criteria (mean age 28.5, SD = 7.82 years). The control group consisted of 10 male violent offenders (mean age of 26.7, SD = 6.5 years) whom not fulfil the criteria of the DSM-IV-R for any diagnosis of personality disorder.

The psychiatric diagnosis was made using clinical and forensic histories of all subjects, which included personal history, education, drug use, mental status, results of structured clinical interview for DSM-IV-R and psychometric tests performed by forensics psychiatrist and psychologist. None of the offenders had a history of major traumatic brain injury and all scored within the range of normal intelligence, measured by the Wechsler Adult Intelligence Scale-Revised (WAIS-R). The criteria for ASPD, included a behavioural pattern that begins before age 15 and comprised at least three of the following behaviours: repeated criminal acts, deceitfulness, impulsiveness, repeated fights or assaults, disregard for the safety of others, irresponsibility and lack of remorse. No subjects were taking any medication at the time of testing.

2.2. EEG procedure

A 21-channel digital EEG equipment from Neuronic SA (RAPTOR 26, Cuba) and an IBM compatible computer were used in the acquisition and storage of EEG data. EEG was recorded from 19 electrode sites (Fp1, Fp2, Fz, F3, F4, F7, F8, Cz, C3, C4, T3, T4, T5, T6, Pz, P3, P4, O1 and O2) according to the International 10–20 system, using surface electrodes referenced to linked ears. Impedance was kept below 5 kV. EEG was amplified by 10,600, with a bandpass at 0.5–30 Hz and sampled through a 12-bit analogue-to-digital converter at 200 Hz.

Electrodes were fitted while subjects were familiarised with the testing equipment and procedure. EEG record was carried out in a quiet, air-conditioned room with the experimenter and recording equipment present. All subjects were instructed to relax and to remain still during testing to minimise artefacts produced by ocular movements, and to avoid excessive blinking. During the recording the subjects were awake with eyes closed, lying on the bed.

Eight to ten minutes of EEG with closed eyes, 2 min of open eyes, 3 min of hyperventilation, 2 min of recuperation were obtained from each subject. In this paper only closed eyes EEG data will be presented.

2.3. Visual assessment of the EEG

Longitudinal and transverse bipolar montages were used for off-line EEG interpretation. The EEG was considered NORMAL if it had an adequate organization of the background activity (according to the subject's age), well defined spatial differentiation, rhythmical alpha activity and absence of paroxysmal activity. The SLOW EEG subgroup was characterized by the presence of persistent non-rhythmic theta–delta slow waves. PAROXYSMAL category included the EEG with activity such as spikes, sharp wave and spike and wave. EEGs with both types of previously described abnormalities were considered in the SLOW and PAROXYSMAL category. Ratios and percentages in all categories were calculated.

2.4. Quantitative EEG analysis (QEEG)

Tracings were visually inspected and edited off-line in order to eliminate epochs with movement artefacts, eye blinking, muscle activity, or drowsiness. For each subject 20–24 EEG segments (without artefact) of 2.56 s at the closed eyes state were selected. Spectral analysis using Fast Fourier Transform (FFT) was carried out in order to obtain the cross-spectral matrixes estimation in all individual records.[33,34] Cross-spectral matrices were calculated for every 0.39 Hz, from 0.78 to 19.53 Hz. All QEEG analysis was made on monopolar leads (linked ears used as reference).

2.5. Statistical analysis

Both experimental and control groups were compared with Cuban normative database by using the *Z* transform.[35] This normative database was constructed from the EEG of 211 normal subjects (105 males, 106 female) and covers an age range from 5 to 97 years. Normative coefficients were obtained by carrying out a polynomial regression with age of each log spectral value. Normalized values, expressed as the number of standard deviations from the mean of the norm, were calculated for every frequency and electrode and stored as a "*Z* spectrum"[35]. Factors like age might affect EEG data by increasing inter-individual variability.[36] The use of normalized values for statistical analysis eliminates these effects that, otherwise, should have been taken into account for comparisons between the groups.

Z values of power for each frequency of both groups were compared by a non-parametric combination of permutation tests.[37,38] This technique allows a distribution-free analysis of the data, and also controls for type I errors, while permitting multiple comparisons in order to detect significant differences in frequencies and electrodes between the groups. Analyses were performed for all measures using a specific software system developed to accomplish it. The level of significance was 0.05 in all cases.

3. Results

3.1. Visual inspection

Visual analyses of rest EEG revealed that 10 violent offenders (42.6%) had disorganization of the background activity, with amplitude of medium voltage range and alpha rhythm attenuation, sometimes barely incipient. Eight of them met ASPD criteria.

Table 1 presents details of the EEG visual analysis results. In both groups results were very similar; SLOW EEG was the category with most subjects in it (around 70%). It was followed by NORMAL EEG (around 20%). Only three subjects of both groups belong to the other two categories. Comparison by means of a Pearson Chi square test found no significant differences between the two

Table 1
Classification of the subject's EEG in both groups by visual inspection

Group	Normal	Slow	Paroxysmal	Slow and paroxysmal
Experimental	3 (16.7%)	13 (72.2%)	1 (5.6%)	1 (5.6%)
Control	2 (20%)	7 (70%)	1 (10%)	0 (0%)

groups for the presence of EEG abnormalities by visual inspection in these four categories ($X^2 = 0.78$, df = 3, $p = 0.85$). Nevertheless, it should be said that in subjects of the ASPD group the amount of slow waves is greater when editing the EEGs than in the control group.

The total amount of abnormal EEGs were of 15 (83.3%) in the experimental group and 8 (80%) in the control group, 23 (82.1%) of the whole sample.

Table 2 shows the topographical distribution of the EEG abnormalities found in both groups. The presence of the EEG abnormality in all recorded brain regions (widespread) was the most frequent localization. Frontal lobe was the single brain region most affected by slow EEG alterations in both groups. A comparison using the Pearson Chi square was made taking into consideration only the SLOW category (due to the fact that it was the EEG abnormality most frequent in both groups) and comparing only widespread vs. frontal localizations. There were no groups differences between these topographical distributions of slow EEG activity ($X^2 = 0.33$, df = 1, $p = 0.57$).

Widespread abnormalities were found in 8 (44.4%) of the experimental group and in 5 (50%) of the control group, 13 (46.4%) of the subjects of both groups taken together.

3.2. Quantitative EEG analysis

Fig. 1 shows the results of the comparison of narrow band spectral frequency values of the ASPD group vs. non-ASPD group using the permutation test. Statistical significant differences were found in:

- Increased delta energy (frequency range 0.78–3.5 Hz) at T3, C4 and P4.
- Increased theta energy (frequency range 4.30–6.64 Hz) at F3, F7 and Pz.
- Increased beta energy (frequency range 14.89–18.75 Hz) at C3, C4, Cz, F4 and P3.
- Decreased alpha energy (frequency range 8.20–10.55 Hz) at T3, C4, PZ, T4 and T5.

In summary the experimental group when compared with the control one basically has an increase of delta at left mid temporal and right central leads, increased theta energy at left frontal region, a decrease of alpha energy at right centro-temporal and left temporal derivations and an excess of beta energy at the central part of the head.

Table 2
EEG abnormalities topographic distribution

Abnormality	Frontal		Temporal		Widespread	
	Experimental group	Control group	Experimental group	Control group	Experimental group	Control group
Slow	5 (27.7%)	2 (20%)	1 (5.6%)	0 (0%)	7 (38.9%)	5 (50%)
Paroxysmal	1 (5.6%)	0 (0%)	0 (0%)	1 (10%)	0 (0%)	0 (0%)
Slow and paroxysmal	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (5.6%)	0 (0%)

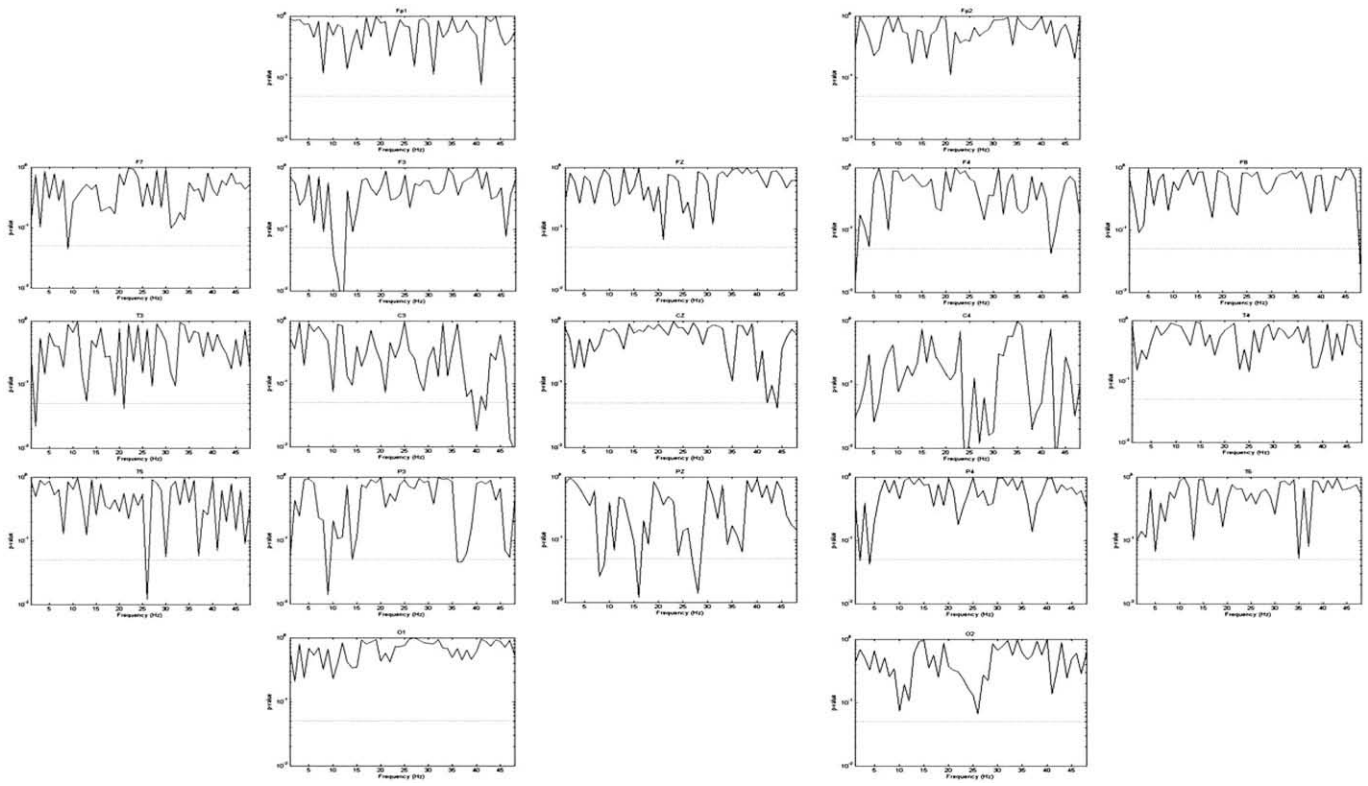

Fig. 1. Comparison of the *Z* values for each frequency component (narrow band spectral frequency analysis) between subjects of the ASPD group versus subject of the non-ASPD group by means of a permutation *t*-test. The scale of the abscissa axis indicates the 48 frequencies (in Hz) of the spectral analysis and the ordinate axis the *t* values expressed in a logarithm scale. Each panel represents an electroencephalographic derivation. Discontinuous lines indicate, where the probability is equal to a signification level of 0.05, significant values are below the lines.

4. Discussion

Widespread and frontal slow wave presence were the localizations and abnormality more frequently found in both groups by visual inspection of the EEGs. Also, disorganization of the background activity was found in subjects of both groups. There were no statistical differences between the experimental and control groups for the presence of abnormalities found neither for the localization of the increased slow wave activity.

Previous research evaluating groups of criminal subjects has reported increased slow waves (delta and theta) activity by visually examining the EEG record.[39–42] Fishbein et al. described that aggressive subjects had more delta and less alpha activity in the spontaneous EEG.[43] All these findings support the presence of CNS abnormalities in violent subjects. None of these works include the diagnosis of ASPD in the subjects studied, and the common conduct characteristic among them is the violent behaviour. Our results are similar to the previously mentioned findings, if it is consider that, although more evident in the ASPD group, the slow wave presence and the decrease of the alpha activity is also present in the control group (violent subjects without the diagnosis of ASPD) and that subjects fulfilling the criteria for ASPD diagnosis should be present in these samples used in previous published work in this area.

Mednick et al. made a review of the studies in which the EEG was evaluated in violent offenders and conclude that there is a high prevalence of EEG abnormalities in violent criminals (ranging from 25% to 50%), especially in recidivistic offenders.[28] The prevalence we found is higher, reaching the 82.1% when considering subjects of both experimental and control groups. In this sense Evan and Park have also found an 85% of abnormal electrophysiological findings in a sample of men convicted of murder.[44]

Quantitative EEG analysis extended the results found by the visual evaluation and differentiated both groups. The experimental group has an excess of energy at the slow frequency bands and a decrease of alpha activity when compared with the control one.

Other authors studying violent subject samples using quantitative EEG analysis had described increased delta and decreased alpha activity in the temporal and parieto-occipital regions[29] or increased delta power in the temporal lobes.[31] These results are, in general, similar to ours. The main difference is related to the localization of the EEG abnormalities. In both previously cited papers bipolar derivations to calculate the broad band spectral parameter were used, and in that case it is important to keep in mind that when assessing voltages, the use of bipolar montages of electrodes may be misleading, since the primary information they provide are related to voltage gradients between the two recording electrodes. For that reason, abnormal EEG activity common to two recording leads used to obtain a bipolar one is been eliminated. Moreover, the diagnosis of ASPD was not used in none of these two articles.

In this sense, Lindberg et al. in 2005 evaluated 16 men with a history of recurrent violent acts, with the diagnosis of ASPD and used quantitative EEG analysis.[45] They found a decrease of alpha power and an increase of theta and delta powers in the waking EEG mostly in occipital regions. Once again, they calculated the broad band spectral parameters from bipolar lead, which is an essential difference with our work. It is curious the fact that in this study all EEG records were classified to be within normal limits by visual inspection, which is a very surprising finding not common in violent offenders samples.

Control group in our study had less severe types of abnormalities. So it is logically to assume that the EEG abnormalities are more in relation with the violent behaviour than with the diagnosis of ASPD. Topographical localizations of abnormalities in our subjects suggest the possible dysfunction of temporal–central regions. The larger amount of abnormalities and the higher severity found in the experimental group in relation to the control one (especially in the quantitative EEG analysis) is a clear evidence that this group of subjects has their brain with more anatomical and/or functional alterations in brain regions, often associated with regulating socially appropriate behaviours. The temporal lobes are involved in affective regulation, sexual behaviour, audition, and speech perception. The anterior–inferior portion of the temporal lobes includes aspects of the limbic system, which is a set of structures, including the amygdala, hippocampus, septum, and cingulate gyrus. Learning and memory, motivation, and emotion regulation are among the most important functions of the limbic system. Abnormalities within the anterior–inferior regions of the temporal lobes are associated with violence.[4,7]

Results of this research may serve as support to the growing evidence of brain dysfunction underlying the diagnosis of ASPD in subject authors of crimes. The use of non-invasive technology like the EEG appears to allow accurate identification of substantial CNS abnormalities. Thus, the visual and quantitative analysis of the resting EEG may continue to be an important tool in the neurological assessment of personality disorder subject.

One aspect that should be emphasized is that some subjects with violent behaviour have a normal EEG record. This fact has been described in every published paper on this matter. So, one person could be an aggressive and violent subject with a normal EEG, and although having a normal EEG does not exclude pathology with its genesis in a dysfunction of the Central Nervous System, it is clear that the presence of slow EEG activity is not a pathognomonic sign of violent behaviour. It could be reflecting a comorbility situation very frequent in this group of subjects.

Trying to explain the differential patterns that emerge from the experimental and control groups when using the QEEG are difficult and maybe unnecessary. The ASPD diagnosis is based on the presence of a number of maladaptive behaviours or mental states identified from a larger set, it is by definition a heterogeneous group, and consequently it lacks specificity. This fact limits the investigation of specific causal factors. Being a heterogeneous group, logically the pattern of abnormalities found in this work should reflect the mean of abnormalities present in the subjects that compose this particular sample. This could explain, at least partially, the discrepancies of results published in this area. It is necessary to reach a more precise specification of the adult antisocial behaviour in order to design a research work more adequately and efficiently to understand the underlying affected process.

Conflict of Interest

None declared.

Funding

No funding.

Ethical Approval

No ethical approval is needed as it is a short report.

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